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WILSON SONSINI GOODRICH & ROSATI
650 PAGE MILL ROAD
PALO ALTO, CA 94304-1050

EXAMINER	
OGUNBIYI, OLUWATOSIN A	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/537,583	VOUSDEN, KATHERINE ANN	
	Examiner	Art Unit	
	OLUWATOSIN OGUNBIYI	1645	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *28 August 2008*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,8,14-16 and 18-35* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,8 and 23-35* is/are rejected.

7)☒ Claim(s) *8* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *9/22/08*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

RESPONSE TO AMENDMENT

The amendment filed 8/28/08 has been entered into the record. Claims 2-7, 9-13 and 17 have been cancelled. Claims 1, 8, 14-16 and 18-35 are pending. Claims 1, 8, and 23-35 are under examination.

Information Disclosure Statement

The information disclosure statement filed 9/22/08 has been considered. An initialed copy is enclosed.

Objections/Rejections Withdrawn

The objection to claims 30 and 31 is withdrawn in view of the amendment to the claims.

The rejection of claim 8 and claims 27-29 and 33-35 under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement is withdrawn in view of the amendment filed 8/28/08.

The rejection of claims 8, 27-29 and 33-35 rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement (new matter rejection) is withdrawn upon further consideration.

The rejection of claims 30-32 under 35 U.S.C. 103(a) as being unpatentable over Weinstock et al. US 6,747,137 B1 published Jun. 8, 2004, filed Feb 12, 1999 (cited in previous action) and Onishi et al. Antimicrobial Agents and Chemotherapy, Feb 2000, p. 368-377 (cited in previous action) as applied to claim 1 further in view of Chen et al (The Journal of Biological Chemistry, 1990, vol. 265, p.16221-16224, cited in IDS) is withdrawn in view of the amendment to the claims.

The rejection of claims 8, 27-29 and 33-35 under 35 U.S.C. 103(a) as being unpatentable over Georgopapadakou et al. Expert Opin. Investig. Drugs (2002) 11 (8):1117-1125 in view of Weinstock et al. US 6,747,137 B1 published Jun. 8, 2004, filed Feb 12, 1999 (cited in previous action) and Nakayama et al. Infection and Immunity , Dec. 2000, p. 6712-6719 and Onishi et al (Feb. 2000, Antimicrobial Agents and Chemotherapy p. 368-377, cited previously) is withdrawn in view of the amendment to claim 8. Applicant's arguments with respect to the claims have been considered but are moot in view of the new ground(s) of rejection.

Objections/Rejections Maintained

1) The objection to claim 8 is maintained: In line 8 please insert 'one' after 'said'.

2) The rejection of claim 1 under 35 U.S.C. 103(a) as being unpatentable over Weinstock et al. US 6,747,137 B1 published Jun. 8, 2004, filed Feb 12, 1999 (cited in previous action) in

view of Onishi et al. Antimicrobial Agents and Chemotherapy, Feb 2000, p. 368-377 (cited in previous action) is maintained for reasons made of record in the office action mailed 5/29/08.

In response to applicant's arguments against the references individually, one cannot show nonobviousness by attacking references individually where the rejections are based on combinations of references. See *In re Keller*, 642 F.2d 413, 208 USPQ 871 (CCPA 1981); *In re Merck & Co.,* 800 F.2d 1091, 231 USPQ 375 (Fed. Cir. 1986).

Applicants argues that Weinstock et al does not disclose the CCA1 from *C. albicans* but discloses from *S. cerevisiae*. This is not persuasive. Weinstock et al discloses nucleic acid sequence relating to *C. albicans*. Table 2, discloses the CCA1 of *C. albicans* contig 3807; column 10 of table 2 provides the name of the organism that was identified as having the closest homology match in this case *S. cerevisiae* and column 11 of the table provides the product name and the function. See column 21 of the specification of Weinstock et al for description of the arrangement of table 2. Thus, Weinstock et al discloses *C. albicans* CCA1. Applicant arguments that it is known that not all proteins essential in *S. cerevisiae* are also essential in *C. albicans* and that genome wide identification of essential genes has not been successfully applied to C. albicans is moot in view of the fact that Weinstock et al discloses *C. albicans* CCA1. Weinstock teaches evaluating a compound for ability to bind a *C. albicans* polypeptide of the invention including CCA1 (column 10 lines 9-17) and not limited to only essential genes. Weinstock teaches that compounds which bind can be candidates as activators or inhibitors of the fungal life cycle. Also, CCA1 of *C. albicans* being an essential gene is an inherent property of said gene and it is not a new property that is conferred on the gene by Applicants. Further, the arguments that CET1 and CDC25 are not essential in *C. albicans* despite being essential in *C. albicans* is

not commensurate in scope with the claims as the claims are not drawn to CET1 and CDC 25 but drawn to CCA1.

To the extent that Applicants are implying that the *C. albicans* CCA1 disclosed in Weinstock et al is not the same as the instant *C. albicans* CCA1, it is noted that the instant claims and the specification does not provide a structure of the instant *C. albicans* CCA1, thus other evidence to the contrary e.g. such as a comparison of the sequences, the CCA1 of Weinstock et al is the same as the instant *C. albicans* CCA1.

Applicants argue that Onishi et al does not disclose CCA1 from *C. albicans*. This is not found persuasive. Applicant argues Onishi individually and not in combination with the primary reference. The combination of Weinstock and Onishi is obvious over claim 1 as set forth previously. Although Weinstock et al does not specifically disclose determining the ability of the candidate compound to inhibit CCA1 activity, it is prima facie obvious to one of ordinary skill in the art at the time that the instant invention was made that determining the binding of a compound to protein such as CCA1 to determine whether it is an anti-fungal candidate necessarily involves the determination that the candidate compound inhibits (or does not inhibit) the activity of said protein. See Onishi et al (above) who teach a method for screening or testing candidate anti-fungal compounds *in vitro* by determining that said candidate anti-fungal inhibit the activity of a target *C. albicans* protein. The point of testing whether a compound is an anti-fungal candidate using a direct test of said compound on a fungal protein is to determine whether the compound inhibits the activity of the protein.

As to Hanic-Joyce et al and Weiner et al, it is noted that these references were not used in making any rejections in any combination i.e. the rejection is over Weinstock and Onishi. The

references were cited in light of Applicants arguments that CCA1 of *Candida* was not an essential gene. Hanic-Joyce et al and Weiner et al do not disclose the instant methods disclosed in the claims and were not cited for disclosing any of the instant methods. Thus, the declaration filed on 8/28/08 under 37 CFR 1.131 to 'swear behind' Hanic-Joyce et al has been considered but is ineffective to overcome the instant rejection as the rejection was not made over Hanic-Joyce et al. Also, arguments as to Navarro et al are not sufficient to overcome the instant rejection as Navarro et al is not used in any combination in the instant rejection. The prior office action never asserted that Navarro et al taught *C. albicans* CCA1 and Navarro was not used in a rejection but was cited in response to Applicants arguments. Navarro was cited for the function of CCA1 i.e. for repair of tRNA molecules missing the 3' terminus and is a ubiquitous enzyme that catalyzes the incorporation of CMP and AMP into incomplete tRNA chains.

3) The rejection of claims 23-26 under 35 U.S.C. 103(a) as being unpatentable over Weinstock et al. US 6,747,137 B1 published Jun. 8, 2004, filed Feb 12, 1999 (cited in previous action) and Onishi et al. Antimicrobial Agents and Chemotherapy, Feb 2000, p. 368-377 (cited in previous action) as applied to claim 1 further in view of Chen et al (The Journal of Biological Chemistry, 1990, vol. 265, p.16221-16224, cited in IDS) is maintained.

Applicants arguments against are essentially for the same reasons as set forth above and have been addressed above. Further, Applicants argue that Chen does not disclose *C. albicans* CCA1 and an assay for screening C. albicans CCA1. In response, the combination of Weinstock, Onishi and Chen discloses *C. albicans* CCA1 and a method for screening C. albicans CCA1.

It would have been prima facie obvious to one of ordinary skill in the art to use the enzymatic assay disclosed by Chen et al for determining the activity of CCA1 (tRNA nucleotidyltransferase) in the method of Weinstock et al and Onishi et al as combined. The enzymatic assay for determining the activity of CCA1 is known in the art and it would have been within the skill of the ordinary artisan to adopt or adapt said enzymatic assay for determining the activity of CCA1 in the presence of a candidate anti-fungal compound.

New Rejections Based on Amendment

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

4) Claims 8 and 27-29 and 30-35 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of screening or testing for candidate anti-fungal compounds that impair *Candida albicans* ATP(CTP):tRNA nucleotidyltransferase enzyme (CCA 1) activity-comprising:

a) providing a *C. albicans* cell wherein the cell expresses *Candida albicans* ATP(CTP):tRNA nucleotidyltransferase enzyme (CCA1)under the control of a heterologous promoter;
b) providing one or more candidate compounds;
c) contacting said *Candida albicans* cell(s) with the said or more candidate compounds; d) determining whether the candidate compound inhibits growth or viability of the cell(s); and

> e) determining whether the candidate compound is a CCA1 inhibitor in a growth in a tRNA nucleotidyl transferase assay,
> does not reasonably provide enablement for said method comprising the step determining whether the candidate compound is a CCA1 inhibitor in a growth inhibition assay or binding assay or a translation inhibition assay.

The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with these claims.

In step e, determining whether the candidate compound is a CCA1 inhibitor in a growth inhibition assay will not necessarily determine whether the candidate compound is a CCA1 inhibitor. If negative effects on growth or viability are seen in said cell(s), how does one of skill in the art determine that said negative effect is due to impairment of CCA1 by the candidate compound? The specification does not correlate impairment of growth or viability with the direct impairment with CCA1. The assay described in the specification (i.e. growth inhibition assay of p. 5 lines 22-30) does not provide any guidance or direction as to how to rule out the effects of such compound on other essential genes in said *C. albicans* expressing CCA1. Assessing inhibition of growth does not provide any knowledge about the effect of the compound on CCA1 activity because as mentioned above a compound may have more that one target in said *C. albicans* expressing CCA1. Furthermore, an anti-fungal compound has many different activities (see Ghannoum et al. 1999. Clinical Microbiology Reviews, p. 501-517 for different mode of actions of some anti-fungal compounds, cited in previous office action) and these compounds would inhibit growth but do not have to impair activity of CCA1.

In step e, determining whether the candidate compound is a CCA1 inhibitor in a binding assay, the claims is broadly drawn to any binding assay. The specification does not provide guidance as

to binding assays that determine whether a candidate compound is a CCA1 inhibitor. A compound binding to CCA1 does not necessarily mean that the compound inhibits the activity of CCA1 e.g. a compound can bind to a portion of CCA1 that is not important for the activity of the enzyme. Even in a competitive assay wherein the binding affinity of the candidate compound is compared with that of a known enzyme substrate for CCA1 such as CTP (specification p. 4 lines 11-15), such a binding assay will not give information on the effects of the compound on the activity of the enzyme. There still needs to be a correlation between binding of compound to the enzyme and the effect of binding on the activity of the enzyme.

In step e, determining whether the candidate compound is a CCA1 inhibitor in a translation inhibition assay, there are many components of the translation machinery including ribosomes, transfer RNA, the mRNA and other enzymes including but not limited to amino-acyl-tRNA synthetics that attach the correct amino acid to each tRNA (see review of protein translation in Molecular Cell Biology, Scientific American Books, New York 1986, pages 107-123). Thus, a candidate compound that inhibits translation does not provide any knowledge as to whether the compound inhibits the activity of the CCA1 enzyme as there are many components available in the translation machinery that the compound can inhibit.

The specification is however enabling for the instant method wherein step e is drawn to determining whether the candidate compound is a CCA1 inhibitor in a tRNA nucleotidyl transferase assay. CCA1 (ATP (CTP): tRNA nucleotidyltransferase) also known amongst many synonyms as tRNA nucleotidyltransferase or CCA-adding enzyme is a ubiquitous enzyme that catalyzes the incorporation of CMP and AMP into incomplete tRNA chains (see

office action mailed 12/22/06 for description of CCA1). Thus, tRNA nucleotidyl transferase assay can directly determine whether the candidate compound identified through steps a-d, inhibits the activity of CCA1.

In view of the above, the specification is enabling for a method of screening or testing for candidate anti-fungal compounds that impair *Candida albicans* ATP(CTP):tRNA nucleotidyltransferase enzyme (CCA 1) activity-comprising:

a) providing a *C. albicans* cell wherein the cell expresses *Candida albicans* ATP(CTP):tRNA nucleotidyltransferase enzyme (CCA1)under the control of a heterologous promoter;

b) providing one or more candidate compounds;

c) contacting said *Candida albicans* cell(s) with the said or more candidate compounds;

d) determining whether the candidate compound inhibits growth or viability of the cell(s); and

e) determining whether the candidate compound is a CCA1 inhibitor in a growth in a tRNA nucleotidyl transferase assay

but does not reasonably provide enablement for said method comprising the step determining whether the candidate compound is a CCA1 inhibitor in a growth inhibition assay or binding assay or a translation inhibition assay.

5) Claims 8, 27-29 and 30-35 are rejected under 35 U.S.C. 103(a) as being unpatentable over Weinstock et al. US 6,747,137 B1 published Jun. 8, 2004, filed Feb 12, 1999 (cited in

previous action) in view of Georgopapadakou et al. Expert Opin. Investig. Drugs (2002) 11 (8):1117-1125 and Nakayama et al. Infection and Immunity , Dec. 2000, p. 6712-6719 and Onishi et al (Feb. 2000, Antimicrobial Agents and Chemotherapy p. 368-377, cited previously) and Chen et al (The Journal of Biological Chemistry, 1990, vol. 265, p.16221-16224, cited in IDS).

The claims are drawn to a method of screening or testing for candidate anti-fungal compounds that impair *Candida albicans* ATP(CTP):tRNA nucleotidyltransferase enzyme (CCA 1) activity-comprising:

a) providing a *C. albicans* cell wherein the cell expresses *Candida albicans* ATP(CTP):tRNA nucleotidyltransferase enzyme (CCA1)under the control of a heterologous promoter;

b) providing one or more candidate compounds;

c) contacting said *Candida albicans* cell(s) with the said or more candidate compounds;

d) determining whether the candidate compound inhibits growth or viability of the cell(s); and

e) determining whether the candidate compound is a CCA1 inhibitor in a growth in a tRNA nucleotidyl transferase assay.

Weinstock et al teach a method of screening test compounds for anti-fungal activity comprising providing a *Candida albicans* target polypeptide sequence such as *Candida albicans* tRNA nucleotidyl transferase also known as CCA1 and contacting a test compound with said CCA 1 and selecting those which bind to said target sequence as anti-fungal candidates (See column 10 lines 9- 17 and 28-45, column 20 lines 46-67 to column 21 lines 1-54 (for description of table 2 column 587-588 contig3807 which discloses *Candida albicans* CCA1). Weinstock teaches that the assays can be performed *in vivo*.

Weinstock does not teach a method for screening or testing candidate antifungal compounds that impair said CCA1 activity comprising a) providing a C. *albicans* cell wherein the cell expresses *Candida albicans* ATP(CTP):tRNA nucleotidyltransferase enzyme (CCA1) under the control of a heterologous promoter; b) providing one or more candidate compounds; c) contacting said *Candida albicans* cell(s) with said or more candidate compounds; d) determining whether the candidate compound inhibits growth or viability of the cell(s); and e) determining whether the candidate compound is a CCA1 inhibitor in a tRNA nucleotidyl transferase assay.

Georgopapadakou et al teach a cell based screening for candidate inhibitors of a *C. albicans* enzyme using a *C. albicans* tetracycline inducible/regulatable promoter system which comprises providing said *C. albicans* expressing said enzyme under said tetracycline inducible/regulatable promoter (p. 1121 column 1 last paragraph).

Nakayama et al teach said tetracycline regulatable system of gene expression in *C. albicans* which uses a tetracycline inducible heterologous tet promoter (see p. 6712 column 1 to column 2, p. 6713 column 1 and column 2 – materials and methods). Nakayama et al teach inducible gene expression in the absence of doxycycline or tetracycline (p. 6712 column 2 first incomplete paragraph and p. 6715 first column) and teach repression of the promoter in the presence of tetracycline or doxycycline thus resulting in inhibition of gene expression ((p. 6712 column 2 first incomplete paragraph and p. 6715 first column to second column).

Onishi et al teaches a cell based screen for antifungal activity of several compounds (which putatively inhibit a fungal enzyme) by a growth inhibition assay on *C. albicans* (page 369

column 1 materials and methods and table 1) and then the compounds were evaluated to determine whether said compounds were direct inhibitors of said enzyme by measuring the enzyme's activity in the presence of said compounds (page 370 column 2 first full paragraph, page 373 column 1 – 2 and table 4).

Chen et al teach an assay for measuring tRNA nucleotidyltransferase activity (aka CCA1) i.e. a tRNA nucleotidyl transferase assay which uses a labeled/radiolabeled nucleotide (3H-CTP).

It would have been prima facie obvious to one of ordinary skill in the art at the time the instant invention was made to screen for candidate compounds that impair the activity of *C. albicans* CCA1 which is disclosed by Weinstock et al using an *in vivo* method such as a cell based screening as taught by Georgopapadakou et al and Nakayama et al. It would have been obvious to express CCA1 under the tetracycline inducible/regulatable promoter system i.e. the tet promoter in *C. albicans* to screen for inhibitors of a *C. albicans CCA1* enzyme as such a cell based screening method for inhibitor of *C. albicans* enzymes is known in the art (Georgopapadakou et al and Nakayama et al) and it would have been prima facie obvious to substitute one enzyme for another in such a screening method to determine whether a candidate compound inhibits growth of *C. albicans* cells expressing said enzyme.

It is obvious that said cell based screening method will involve the further determination of the effect of said compound on the growth and viability of said *C. albicans* (since the method is directed at cell based screening for an antifungal compound) and it would be prima facie obvious to compare the growth or viability of said *C. albicans* when the tet promoter is repressed (no CCA1 expression) as a control to determine any differences in the effect of compounds on the growth or viability of *C. albicans* in the presence or absence of CCA1 expression.

Further it is obvious that validation tests are further carried out to determine that said candidate antifungal compound inhibits enzyme activity in the instant case CCA1 activity directly. This is because Onishi et al teaches that after a cell based screen for antifungal activity of several compounds (which putatively inhibit an enzyme's activity) by a growth inhibition assay (page 369 column 1 materials and methods and table 1) and the compounds are further evaluated to determine whether said compounds were direct inhibitors of the enzyme by measuring the enzyme's activity in the presence of said compounds (page 370 column 2 first full paragraph, page 373 column 1 – 2 and table 4). One would have a reasonable expectation of success because assays for determining the activity of *C. albicans* CCA1 because assays for measuring the activity of CCA1 i.e. tRNA nucleotidyl transferase assay which uses a labeled/radiolabeled nucleotide (3H-CTP),were known in the art (see Chen et al) as of the time of the making of the instant invention. It is reasonable for one of skill in the art to adapt a known assay for measuring an enzyme/s activity for measuring the activity of the same enzyme, albeit from a different fungus.

Status of Claims

Claims 1, 8, and 23-35 are under examination. No claims allowed.

Conclusion

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to OLUWATOSIN OGUNBIYI whose telephone number is 571-272-9939. The examiner can normally be reached on M-F 8:30 am- 5 pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Robert Mondesi can be reached on 571-272-0956. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Oluwatosin Ogunbiyi/
Examiner, Art Unit 1645

/Robert B Mondesi/
Supervisory Patent Examiner,
Art Unit 1645